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著者名(英)	Turner,R.James/D.,Ph.
journal or publication title	東日本歯学雑誌
volume	11
number	2
page range	230
year	1992-12-31
URL	http://id.nii.ac.jp/1145/00007799/

Mechanisms of Fluid Secretion in Salivary Glands : Energetics and Regulation

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Based on recent studies with rat and rabbit salivary glands three mechanisms have been proposed for primary fluid secretion by salivary acini. These mechanisms are all based on the concept that the acinar cells first secrete salt via active anion transport, with water following salt osmotically. Two of these models involve active Cl^- secretion via an apical Cl^- channel with basolateral Cl^- entry via either a Na^+-K^+-$2Cl^-$ cotransporter (Model 1) or a Cl^-/HCO_3^- exchanger (Model 2). In Model 3 active HCO_3^- secretion occurs via an apical HCO_3^- channel, possibly the same channel responsible for Cl^- secretion. In Models 2 and 3 the intracellular acid load resulting from acinar HCO_3^- loss (via Cl^-/HCO_3^- exchange or the apical HCO_3^- channel, respectively) is buffered by a Na^+/H^+ exchanger. Interestingly, at least in the parotid and submandibular glands of the rat and rabbit, these three models do not appear to be alternate explanations for the fluid secretory process, but rather mechanisms which operate concurrently in the same gland and quite possibly in the same acinar cells. At the present time the physiological significance of this multiplicity of secretory mechanisms is uncertain, however, there is good evidence that the relative contributions of each of the above models to fluid secretion varies from species to species and from gland to gland. Recent work from our laboratory has also shown that several of the ion transport systems involved in salivary fluid secretion are tightly regulated by secretagogues. Interference with these regulatory events by certain clinical agents may account for the dry mouth and dry eyes experienced by some patients taking these medications.

平成4年9月1日　**(口腔生化学)**